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**********STN Columbus **********
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FILE 'HOME' ENTERED AT 18:09:14 ON 22 JUN 2000

=> file medline

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                                    ENTRY SESSION
FULL ESTIMATED COST                   0.15     0.15

FILE 'MEDLINE' ENTERED AT 18:09:28 ON 22 JUN 2000

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 Basic Index. See HELP SFIELDS for details.

 THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY
AND ACCURATE
 SUBSTANCE IDENTIFICATION.

=> s zinc finger and dna binding/ab,bi

      43953 ZINC
      23185 FINGER
       3581 ZINC FINGER
            (ZINC(W)FINGER)
     555395 DNA/BI
     504902 BINDING/BI
    5335587 AB/FA
      21855 DNA BINDING/AB
            ((DNA(W)BINDING)/BI (L) AB/FA)
     555395 DNA/BI
     504902 BINDING/BI
      52599 DNA BINDING/BI
            ((DNA(W)BINDING)/BI)
L1     2136 ZINC FINGER AND DNA BINDING/AB,BI

=> s l1 and polypeptide/ab,bi

      55564 POLYPEPTIDE/BI
    5335587 AB/FA
      49330 POLYPEPTIDE/AB
            (POLYPEPTIDE/BI (L) AB/FA)
      55564 POLYPEPTIDE/BI
L2       87 L1 AND POLYPEPTIDE/AB,BI

=> s l2 and methyltransferase/ab,bi

       9693 METHYLTRANSFERASE/BI
    5335587 AB/FA
       5361 METHYLTRANSFERASE/AB
            (METHYLTRANSFERASE/BI (L) AB/FA)
       9693 METHYLTRANSFERASE/BI
L3        0 L2 AND METHYLTRANSFERASE/AB,BI

=> s cpg/ab,bi

       3392 CPG/BI
    5335587 AB/FA
       3148 CPG/AB
            (CPG/BI (L) AB/FA)
       3392 CPG/BI
L4     3392 CPG/AB,BI

=> s l4 and methyltransferase/ab,bi

       9693 METHYLTRANSFERASE/BI
    5335587 AB/FA
       5361 METHYLTRANSFERASE/AB
            (METHYLTRANSFERASE/BI (L) AB/FA)
       9693 METHYLTRANSFERASE/BI
L5      209 L4 AND METHYLTRANSFERASE/AB,BI

=> s l5 and zinc/ab,bi

      43953 ZINC/BI
    5335587 AB/FA
      23697 ZINC/AB
            (ZINC/BI (L) AB/FA)
      43953 ZINC/BI
L6        6 L5 AND ZINC/AB,BI

=> d l- bib ab

YOU HAVE REQUESTED DATA FROM 6 ANSWERS -
CONTINUE? Y/(N):y


L6  ANSWER 1 OF 6  MEDLINE
AN  1999299250    MEDLINE
DN  99299250
TI  Multipoint analysis of human chromosome 11p15/mouse distal
chromosome 7:
    inclusion of H19/IGF2 in the minimal WT2 region, gene specificity
of H19
    silencing in Wilms' tumorigenesis and methylation
hyper-dependence of H19
    imprinting.
AU  Dao D; Walsh C P; Yuan L; Gorelov D; Feng L; Hensle T;
Nisen P; Yamashiro
    D J; Bestor T H; Tycko B
CS  Department of Pathology and Institute for Cancer Genetics,
Columbia
    Univesity College of Physicians and Surgeons, New York, NY
10032, USA.
SO  HUMAN MOLECULAR GENETICS, (1999 Jul) 8 (7) 1337-52.

    Journal code: BRC. ISSN: 0964-6906.
CY  ENGLAND: United Kingdom
DT  Journal; Article; (JOURNAL ARTICLE)
LA  English
FS  Priority Journals
EM  199910
AB  WT2 is defined by maternal-specific loss of heterozygosity
(LOH) on
    chromosome 11p15.5 in Wilms' tumors (WTs). The imprinted H19
gene, in this
    region, is silenced and hypermethylated in most WTs, and this is
linked to
    pathological biallelic expression of IGF2. However, H19 and IGF2
lie
    within a larger imprinted domain, and the gene specificity of H19
    epimutation has been a persistent question. To address this, we
assessed
    LOH, gene expression and DNA methylation at multiple sites in
and around
    the imprinted domain. LOH mapping showed that the entire
domain, including
    IGF2/H19, is within the minimal WT2 region. Genes within the
domain,
    including IPL/TSSC3/BWR1C,
IMPT1/ORCTL2/BWR1A/TSSC5, KvLQT1/KCNA9 and
    TAPA1/CD81, as well as the ***zinc*** finger gene
ZNF195/ZNFP104 near
    the centromeric border, were expressed persistently in many WTs.
DNA
    hypermethylation was not detected with 5" upstream probes for
IPL, IMPT1,
    KvLQT1 and ZNF195 in WTs or WT-associated kidneys. Fully
developed WTs
    showed variable hypomethylation at an imprinted ***CpG***
island in a
    KvLQT1 intron, but this was only complete in the cases with LOH
and was
    not observed in pre-neoplastic WT-associated kidneys with H19
epimutation.
    Analysis of the corresponding region of mouse chromosome 7 using
    ***methyltransferase*** -hypomorphic mice showed that the
H19 imprint was
    fully erased, but that the allelic bias at Ipl, Impt1, p57 Kip2 and, to
a
    lesser extent, Kvlqt1, persisted. Pre-existing massive allelic
asymmetry
    for DNA methylation and hyper-dependence of transcription on
methylation
    status may underlie the mechanism of gene-specific silencing of
```

H19 in
Wilms' tumorigenesis.

L6 ANSWER 2 OF 6 MEDLINE
AN 1998061079 MEDLINE
DN 98061079
TI Cytosine methylation targetted to pre-determined sequences [letter].
AU Xu G L; Bestor T H
NC GM00616 (NIGMS)
AI40021 (NIAID)
SO NATURE GENETICS, (1997 Dec) 17 (4) 376-8.
Journal code: BRO. ISSN: 1061-4036.
CY United States
DT Letter
LA English
FS Priority Journals
EM 199803
EW 19980301

L6 ANSWER 3 OF 6 MEDLINE
AN 97392433 MEDLINE
DN 97392433
TI DNA (cytosine-5)-methyltransferases in mouse cells and tissues. Studies
with a mechanism-based probe.
AU Yoder J A; Soman N S; Verdine G L; Bestor T H
CS Department of Genetics and Development, College of Physicians and Surgeons
of Columbia University, New York, NY 10032, USA.
SO JOURNAL OF MOLECULAR BIOLOGY, (1997 Jul 18) 270 (3) 385-95.
Journal code: J6V. ISSN: 0022-2836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199710
EW 19971005
AB The mechanisms that establish and maintain methylation patterns in the
mammalian genome are very poorly understood, even though perturbations of
methylation patterns lead to a loss of genomic imprinting, ectopic X
chromosome inactivation, and death of mammalian embryos. A family of
sequence-specific DNA methyltransferases has been proposed to be
responsible for the wave of de novo methylation that occurs in the early
embryo, although no such enzyme has been identified. A universal
mechanism-based probe for DNA (cytosine-5)-methyltransferases was used to
screen tissues and cell types known to be active in de novo methylation
for new species of DNA ***methyltransferase*** . All identifiable de
novo ***methyltransferase*** activity was found to reside in Dnmt1. As
this enzyme is the predominant de novo ***methyltransferase*** at all
developmental stages inspected, it does not fit the definition of
maintenance ***methyltransferase*** or hemimethylase. Recent genetic
data indicate that de novo methylation of retroviral DNA in embryonic stem
cells is likely to involve one or more additional DNA methyltransferases.
Such enzymes were not detected and are either present in very small
amounts or are very different from Dnmt1. A new method was developed and
used to determine the sequence specificity of intact Dnmt1 in whole-cell
lysates. Specificity was found to be confined to the sequence 5'-
CpG -3'; there was little dependence on sequence context or density
of ***CpG*** dinucleotides. These data suggest that any
sequence-specific de novo methylation mediated by Dnmt1 is either under
the control of regulatory factors that interact with Dnmt1, or is cued by
alternative secondary structures in DNA.

L6 ANSWER 4 OF 6 MEDLINE
AN 97334329 MEDLINE
DN 97334329
TI Mouse DNA ***methyltransferase*** (MTase) deletion mutants that retain
the catalytic domain display neither de novo nor maintenance methylation
activity in vivo.
AU Zimmermann C; Guhl E; Graessmann A
CS Institut fur Molekularbiologie und Biochemie Freie Universitat Berlin,
Germany.
SO BIOLOGICAL CHEMISTRY, (1997 May) 378 (5) 393-405.
Journal code: CK4. ISSN: 1431-6730.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199712
EW 19971201
AB The mammalian genome encodes a DNA cytosine-5-***methyltransferase***
(MTase) of about 170 kDa that is apparently responsible for both de novo
and maintenance methylation at ***CpG*** sites. Both methylation
activities have to be regulated accurately to ensure correct developmental
and cell type-specific gene activity. Distorted DNA methylation patterns
have been associated with cell aging and diseases such as cancer and
fragile X syndrome. Structural and functional in vitro studies of the
mouse MTase have indicated that the enzyme has both a regulatory and a
catalytic region located in the N-terminal and C-terminal parts of the
protein, respectively. The regulatory region includes the nuclear
localization signal (NLS), the sequence for DNA targeting and the
Zn-binding domain. The catalytic domain carries the ten consensus sequence
motifs specific for all known pro- and eukaryotic DNA cytosine-5-
methyltransferases. In an attempt to separate regulatory and catalytic
functions of the enzyme in vivo, we have tested various deletion mutations
by means of transient and stable cell transfection experiments. Expression
of the transgenes, all of which retained the C-terminal catalytic domain,
was monitored by immunofluorescence staining, Northern blot analysis and
SDS gel electrophoresis. Despite high levels of transgene expression, the
truncated MTase molecules exhibited neither de novo nor maintenance
methylation activity. These findings might indicate that in vivo, an
efficient control mechanism prevents the ectopic activity of the DNA MTase
that is structurally compromised in its N-terminal regulatory region.

L6 ANSWER 5 OF 6 MEDLINE
AN 97121258 MEDLINE
DN 97121258
TI ***Zinc*** dependent recognition of a human ***CpG*** island
sequence by the mammalian spermatidal protein TP2.
AU Kundu T K; Rao M R
CS Department of Biochemistry, Indian Institute of Science, Bangalore, India.
SO BIOCHEMISTRY, (1996 Dec 10) 35 (49) 15626-32.
Journal code: A0G. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199703
AB Rat spermatidal protein TP2 is a ***zinc*** metalloprotein with two
atoms of ***zinc*** coordinated to cysteine and histidine residues and
condenses alternating GC copolymer preferentially in a ***zinc***
dependent manner [Kundu, T. K., & Rao, M. R. S. (1995) Biochemistry
34,5143-5150]. In the present study, we have used a 40-mer oligonucleotide
containing a human ***CpG*** island sequence to study its

interaction
with TP2 by gel mobility shift assays. A specific complex was observed in
the presence of poly(dI).poly(dC). Preincubation of TP2 with 10 mM EDTA or
1 mM 1, 10-o-phenanthroline inhibited the complex formation by more than
90%. Competition experiments with various polynucleotides revealed the
following order of efficiency: poly(dG-dC).poly(dG-dC) > cold homologous
oligonucleotide > poly(dA-dT).poly(dA-dT). Homoduplexes poly(dG).poly(dC)
and poly(dA).poly(dT) had no effect on the complex formation. Chromomycin
A3, a GC minor groove binding drug, inhibited the complex formation.
Methylation of the ***CpG*** doublet within the ***CpG*** island
sequence by SssI methylase (***CpG*** methylase) completely abolished
the complex formation. Methylation of G at the N-7 position with dimethyl
sulfate did not affect the recognition of ***CpG*** island by TP2.
Thus, ***CpG*** islands, widely distributed in the mammalian genome,
may serve as specific loci for initiation of chromatin condensation by TP2
during the later stages of spermiogenesis.

L6 ANSWER 6 OF 6 MEDLINE
AN 96187795 MEDLINE
DN 96187795
TI Analysis of 94 kb of the chlorella virus PBCV-1 330-kb genome: map
positions 88 to 182.
AU Lu Z; Li Y; Que Q; Kutish G F; Rock D L; Van Etten J L
CS Plum Island Animal Disease Center, ARS, USDA, NAA, Greenport, New York
11944-0848, USA.
NC GM-32441 (NIGMS)
SO VIROLOGY, (1996 Feb 1) 216 (1) 102-23.
Journal code: XEA. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK-U42580; GENBANK-U17055; GENBANK-L28919; GENBANK-U18997;
GENBANK-D31818; GENBANK-P14529; GENBANK-P18997; GENBANK-P30320;
GENBANK-P15436; GENBANK-P30315; GENBANK-P28340; GENBANK-P24907;
GENBANK-P12004; GENBANK-P04961; GENBANK-P17070; GENBANK-P11038;
GENBANK-P11926; GENBANK-P07805; GENBANK-P00860; GENBANK-P08432;
GENBANK-P35207; GENBANK-D29641; GENBANK-U20861; GENBANK-P00442;
GENBANK-P00445; GENBANK-P28756; GENBANK-P24705; GENBANK-L35601;
GENBANK-Q06527; GENBANK-U14659; +
EM 199608
AB Analysis of 94 kb of DNA, located between map positions 88 and 182 kb in
the 330-kb chlorella virus PBCV-1 genome, revealed 195 open reading frames
(ORFs) 65 codons or longer. One hundred and five of the 195 ORFs were
considered major ORFs. Twenty-six of the 105 major ORFs resembled genes in
the databases including three chitinases, a chitosanase, three
serine/threonine protein kinases, two additional protein kinases, a
tyrosine protein phosphatase, two ankyrins, an ornithine decarboxylase, a
copper/ ***zinc*** -superoxide dismutase, a proliferating cell nuclear
antigen, a DNA polymerase, a fibronectin-binding protein, the yeast Ski2
protein, an adenine DNA ***methyltransferase*** and its corresponding
DNA site-specific endonuclease, and an amidase. The genes for the 105
major ORFs were evenly distributed along the genome and, except for one
noncoding 1788-nucleotide stretch, the genes were close together.
Unexpectedly, a 900-bp region in the 1788-bp noncoding sequence resembled
a ***CpG*** island.

=> s cpg-specific/ab,bi

3392 CPG/BI
641947 SPECIFIC/BI
5335587 AB/FA
12 CPG-SPECIFIC/AB
((CPG(W)SPECIFIC)/BI (L) AB/FA)
3392 CPG/BI
641947 SPECIFIC/BI
12 CPG-SPECIFIC/BI
((CPG(W)SPECIFIC)/BI)
L7 12 CPG-SPECIFIC/AB,BI

=> s l7 and methyltransferase#/ab,bi

11595 METHYLTRANSFERASE#/BI
5335587 AB/FA
5689 METHYLTRANSFERASE#/AB
(METHYLTRANSFERASE#/BI (L) AB/FA)
11595 METHYLTRANSFERASE#/BI
L8 2 L7 AND METHYLTRANSFERASE#/AB,BI

=> d l- bib ab

YOU HAVE REQUESTED DATA FROM 2 ANSWERS - CONTINUE? Y/(N):y

L8 ANSWER 1 OF 2 MEDLINE
AN 95116326 MEDLINE
DN 95116326
TI The ***CpG*** - ***specific*** methylase SssI has topoisomerase
activity in the presence of Mg2+.
AU Matsuo K; Silke J; Gramatikoff K; Schaffner W
CS Institut fur Molekularbiologie II, Universitat Zurich, Switzerland.
SO NUCLEIC ACIDS RESEARCH, (1994 Dec 11) 22 (24) 5354-9.
Journal code: O8L. ISSN: 0305-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199504
AB A prokaryotic ***CpG*** - ***specific*** methylase from Spiroplasma,
SssI methylase, is now widely used to study the effect of CpG methylation
in mammalian cells, and can processively modify cytosines in CpG
dinucleotides in the absence of Mg2+. In the presence of Mg2+, we found
(i) that the methylation reaction is distributive rather than processive
as a result of the decreased affinity of SssI methylase for DNA, and (ii)
that a type I-like topoisomerase activity is present in SssI methylase
preparations. This topoisomerase activity was still present in SssI
methylase further purified by either SDS-polyacrylamide or isoelectric
focusing gel electrophoresis. We show that methylase and topoisomerase
activities are not functionally interdependent, since conditions exist
where only one or the other enzymatic activity is detectable. The
catalytic domains of SssI methylase and prokaryotic topoisomerases show
similarity at the amino acid level, further supporting the idea that the
topoisomerase activity is a genuine activity of SssI methylase.
Mycoplasmas, including Spiroplasma, have the smallest genomes of all
living organisms; thus, this condensation of two enzymatic activities into
the same protein may be a result of genome economy, and may also have
functional implications for the mechanism of methylation.

L8 ANSWER 2 OF 2 MEDLINE
AN 91162719 MEDLINE
DN 91162719
TI Adenovirus type 2 VAI RNA transcription by polymerase III is

blocked by
sequence-specific methylation.
AU Juttermann R; Hosokawa K; Kochanek-S; Doerfler W
CS Institute of Genetics, University of Cologne, Federal Republic of Germany.
SO JOURNAL OF VIROLOGY, (1991 Apr) 65 (4) 1735-42.
Journal code: KCV. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199106
AB Sequence-specific methylation of the promoter and adjacent regions in
mammalian genes transcribed by RNA polymerase II leads to the inhibition
of these genes. So far, RNA polymerase III-transcribed genes have not been
investigated in depth. We therefore studied methylation effects on the RNA
polymerase III-transcribed VAI gene of adenovirus type 2 DNA. The VAI gene
contains 20 5'-CG-3' dinucleotides, of which 4 (20%) can be methylated by
HpaII (5'-CCGG-3') and HhaI (5'-GCGC-3'). Three of these 5'-CG-3'
sequences are located close to the internal regulatory region of the VAI
segment. An unmethylated, a 5'-CCGG-3'- and 5'-GCGC-3'-methylated, and a
5'-CG-3'-methylated pUC18 construct containing the VAI and VAII regions
were transfected into mammalian cells. In many experiments, an
inactivating effect of 5'-CCGG-3' and 5'-GCGC-3' DNA methylation on the
VAI region was not observed. In contrast, methylation of all 20 5'-CG-3'
sequences in the VAI region by a ***CpG*** - ***specific*** DNA
methyltransferase from Spiroplasma species did interfere with VAI
transcription. Transcription of the VAI- and VAII- and of the
VAI-containing constructs was also shown to be inhibited in an in vitro
cell-free transcription system after the constructs had been methylated at
the 5'-CCGG-3' and 5'-GCGC-3' sequences or at all 5'-CG-3' sequences. When
an oligodeoxyribonucleotide which carried the internal control block A of
the VAI region was methylated at three 5'-CG-3' sequences, the formation
of a complex with HeLa nuclear proteins was abrogated. The results
presented support the notion that the VAI gene transcribed by the
DNA-dependent RNA polymerase III is also inactivated by methylation of the
decisive 5'-CG-3' sequences.

=> s chimeric or chimera/ab,bi

14856 CHIMERIC
10508 CHIMERA/BI
5335587 AB/FA
2241 CHIMERA/AB
(CHIMERA/BI (L) AB/FA)
10508 CHIMERA/BI
L9 22158 CHIMERIC OR CHIMERA/AB,BI

=> s l9 and zinc-finger/ab,bi

43953 ZINC/BI
23185 FINGER/BI
5335587 AB/FA
3326 ZINC-FINGER/AB
((ZINC(W)FINGER)/BI (L) AB/FA)
43953 ZINC/BI
23185 FINGER/BI
3581 ZINC-FINGER/BI
((ZINC(W)FINGER)/BI)
L10 114 L9 AND ZINC-FINGER/AB,BI

=> s l10 and dna binding/ab,bi

555395 DNA/BI
504902 BINDING/BI
5335587 AB/FA
21855 DNA BINDING/AB
((DNA(W)BINDING)/BI (L) AB/FA)
555395 DNA/BI
504902 BINDING/BI
52599 DNA BINDING/BI
((DNA(W)BINDING)/BI)
L11 83 L10 AND DNA BINDING/AB,BI

=> s l11 and cpg?/ab,bi

3610 CPG?/BI
5335587 AB/FA
3357 CPG?/AB
(CPG?/BI (L) AB/FA)
3610 CPG?/BI
L12 1 L11 AND CPG?/AB,BI

=> d bib ab

L12 ANSWER 1 OF 1 MEDLINE
AN 2000122256 MEDLINE
DN 20122256
TI The cell cycle control gene ZAC/PLAGL1 is imprinted--a strong candidate
gene for transient neonatal diabetes.
AU Kamiya M; Judson H; Okazaki Y; Kusakabe M; Muramatsu M; Takada S; Takagi
N; Arima T; Wake N; Kamimura K; Satomura K; Hermann R; Bonthron D T;
Hayashizaki Y
CS CREST, Japan Science and Technology Corporation (JST), Genome Exploration
Research Group, Genomic Sciences Center (GSC), Genome Science Laboratory
and Biogenetic Research Center, Riken Tsukuba Life Science Center,
Ibaraki, Japan.
SO HUMAN MOLECULAR GENETICS, (2000 Feb 12) 9 (3) 453-60.
Journal code: BRC. ISSN: 0964-6906.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200007
EW 20000701
AB We describe a screen for new imprinted human genes, and the identification
in this way of ZAC (***zinc*** ***finger*** protein which regulates apoptosis and cell cycle arrest)/ PLAGL1 (pleomorphicadenoma of
the salivary gland gene like 1) as a strong candidate gene for transient
neonatal diabetes mellitus (TNDM). To screen for imprinted genes, we
compared parthenogenetic DNA from the ***chimeric*** patient FD and
androgenetic DNA from hydatidiform mole, using restriction landmark genome
scanning for methylation. This resulted in identification of two novel
imprinted loci, one of which (NV149) we mapped to the TNDM region of 6q24.
From analysis of the corresponding genomic region, it was determined that
NV149 lies approximately 60 kb upstream of the ZAC / PLAGL1 gene. RT-PCR
analysis was used to confirm that this ZAC / PLAGL1 is expressed only from
the paternal allele in a variety of tissues. TNDM is known to result from
upregulation of a paternally expressed gene on chromosome 6q24. The
paternal expression, map position and known biological properties of ZAC /
PLAGL1 make it highly likely that it is the TNDM gene. In particular, ZAC
/ PLAGL1 is a transcriptional regulator of the type 1 receptor for
pituitary adenylate cyclase-activating polypeptide, which is the most
potent known insulin secretagog and an important mediator of autocrine

control of insulin secretion in the pancreatic islet.

=> file medline embase biosis inpadoc caplus

COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 4.36 4.51

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FILE 'EMBASE' ENTERED AT 18:14:34 ON 22 JUN 2000
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=> s l12

'AB' IS NOT A VALID FIELD CODE
L13 2 L12

=> dup rem l13

PROCESSING COMPLETED FOR L13
L14 2 DUP REM L13 (0 DUPLICATES REMOVED)

=> d 1- bib ab

YOU HAVE REQUESTED DATA FROM 2 ANSWERS - CONTINUE? Y/(N):y

L14 ANSWER 1 OF 2 MEDLINE
AN 2000122256 MEDLINE
DN 20122256
TI The cell cycle control gene ZAC/PLAGL1 is imprinted--a strong candidate
gene for transient neonatal diabetes.
AU Kamiya M; Judson H; Okazaki Y; Kusakabe M; Muramatsu M; Takada S; Takagi
N; Arima T; Wake N; Kamimura K; Satomura K; Hermann R; Bonthron D T;
Hayashizaki Y
CS CREST, Japan Science and Technology Corporation (JST), Genome Exploration
Research Group, Genomic Sciences Center (GSC), Genome Science Laboratory
and Biogenetic Research Center, Riken Tsukuba Life Science Center,
Ibaraki, Japan.
SO HUMAN MOLECULAR GENETICS, (2000 Feb 12) 9 (3) 453-60.
Journal code: BRC. ISSN: 0964-6906.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200007
EW 20000701
AB We describe a screen for new imprinted human genes, and the identification
in this way of ZAC (***zinc*** ***finger*** protein which
regulates apoptosis and cell cycle arrest)/ PLAGL1 (pleomorphicadenoma of
the salivary gland gene like 1) as a strong candidate gene for transient
neonatal diabetes mellitus (TNDM). To screen for imprinted genes, we
compared parthenogenetic DNA from the ***chimeric*** patient FD and
androgenetic DNA from hydatidiform mole, using restriction landmark genome
scanning for methylation. This resulted in identification of two novel
imprinted loci, one of which (NV149) we mapped to the TNDM region of 6q24.
From analysis of the corresponding genomic region, it was determined that
NV149 lies approximately 60 kb upstream of the ZAC / PLAGL1 gene. RT-PCR
analysis was used to confirm that this ZAC / PLAGL1 is expressed only from
the paternal allele in a variety of tissues. TNDM is known to result from
upregulation of a paternally expressed gene on chromosome 6q24. The
paternal expression, map position and known biological properties of ZAC /
PLAGL1 make it highly likely that it is the TNDM gene. In particular, ZAC
/ PLAGL1 is a transcriptional regulator of the type 1 receptor for
pituitary adenylate cyclase-activating polypeptide, which is the most
potent known insulin secretagog and an important mediator of autocrine
control of insulin secretion in the pancreatic islet.

L14 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2000 ACS
AN 1999:725359 CAPLUS
DN 132:48369
TI The promyelocytic leukemia ***zinc*** ***finger*** (PLZF) protein
binds DNA in a high molecular weight complex associated with cdc2 kinase
AU Ball, Helen J.; Melnick, Ari; Shaknovich, Rita; Kohanski, Ronald A.;
Licht, Jonathan D.
CS Derald H. Ruttenberg Cancer Center, Mount Sinai School of Medicine, New
York, NY, 10029, USA
SO Nucleic Acids Res. (1999), 27(20), 4106-4113
CODEN: NARHAD; ISSN: 0305-1048
PB Oxford University Press
DT Journal
LA English
AB A binding site selection from a ***CpG*** island library for the
promyelocytic leukemia ***zinc*** ***finger*** protein (PLZF)
identified two high affinity PLZF binding sites. These sequences also
bound RAR.alpha./PLZF, a fusion protein formed in chromosomal
translocation t(11;17)(q23;q21) assocd. with acute promyelocytic leukemia.
PLZF bound DNA as a slowly migrating complex with an estd. mol. wt. of 600
kDa whose formation was dependent on the POZ/dimerization domain of PLZF.
The PLZF-DNA complex was unable to form in the presence of cdc2
antibodies. A PLZF-cdc2 interaction was further demonstrated by
co-immunopptn. and a biotin-streptavidin pull-down assay. PLZF is a
phosphoprotein and immunoppts. with a cdc2-like kinase activity. The
PLZF-DNA complex was abolished with the addn. of a phosphatase. These
studies suggest that the activity of PLZF, a regulator of the cell cycle,
may be modulated by cell cycle proteins. RAR.alpha./PLZF did not complex
with cdc2, this potentially contributing to its aberrant transcriptional
properties and potential role in leukemogenesis.
RE.CNT 55
RE
(1) Ahmad, K; Proc Natl Acad Sci USA 1998, V95, P12123 CAPLUS
(2) Alcalay, M; Mol Cell Biol 1998, V18, P1084 CAPLUS
(3) Andrews, N; Nucleic Acids Res 1991, V19, P2499 CAPLUS
(5) Boyle, W; Methods Enzymol 1991, V201, P110 CAPLUS
(6) Chang, K; Blood 1992, V79, P554 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> file medline

COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 24.18 28.69

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
SINCE FILE TOTAL
ENTRY SESSION
CA SUBSCRIBER PRICE -0.56 -0.56

FILE 'MEDLINE' ENTERED AT 18:16:29 ON 22 JUN 2000

FILE LAST UPDATED: 16 JUN 2000 (20000616/UP). FILE COVERS 1960 TO DATE.

MEDLINE has been reloaded to reflect the annual MeSH changes made by
the National Library of Medicine for 2000. Enter HELP RLOAD for details.

OLDMEDLINE, data from 1960 through 1965 from the Cumulated Index
Medicus (CIM), has been added to MEDLINE. See HELP CONTENT for details.

Left, right, and simultaneous left and right truncation are available in the
Basic Index. See HELP SFIELDS for details.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE
SUBSTANCE IDENTIFICATION.

=> s zinc-finger binding or zinc-finger dna binding/ab,bi

43953 ZINC
23185 FINGER
504902 BINDING
17 ZINC-FINGER BINDING
(ZINC(W)FINGER(W)BINDING)
43953 ZINC/BI
23185 FINGER/BI
555395 DNA/BI
504902 BINDING/BI
5335587 AB/FA
186 ZINC-FINGER DNA BINDING/AB
((ZINC(W)FINGER(W)DNA(W)BINDING)/BI (L) AB/FA)
43953 ZINC/BI
23185 FINGER/BI
555395 DNA/BI
504902 BINDING/BI
203 ZINC-FINGER DNA BINDING/BI
((ZINC(W)FINGER(W)DNA(W)BINDING)/BI)
L15 217 ZINC-FINGER BINDING OR ZINC-FINGER DNA BINDING/AB,BI

=> s l15 and cpg?/ab,bi

3610 CPG?/BI
5335587 AB/FA
3357 CPG?/AB
(CPG?/BI (L) AB/FA)
3610 CPG?/BI
L16 1 L15 AND CPG?/AB,BI

=> d bib ab

L16 ANSWER 1 OF 1 MEDLINE
AN 92241871 MEDLINE
DN 92241871
TI Structural and functional organization of the HF.10 human zinc finger gene
(ZNF35) located on chromosome 3p21-p22.
AU Lanfrancone L; Pengue G; Pandolfi P P; Salcini A E; Giacomucci A; Longo L;
Donti E; De Luca P; La Mantia G; Pelicci P G; et al
CS Istituto di Clinica Medica I, University of Perugia, Italy..
SO GENOMICS, (1992 Apr) 12 (4) 720-8.
Journal code: GEN. ISSN: 0888-7543.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-S98392; GENBANK-S98395; GENBANK-S98399; GENBANK-S98401;
GENBANK-M86366; GENBANK-M86367; GENBANK-M86371; GENBANK-M86372;
GENBANK-M86373; GENBANK-M86374
EM 199208
AB We report the structural and functional characterization of the HF.10 zinc
finger gene (ZNF35) in normal human cells, as well as a processed
pseudogene. The HF.10 gene spans about 13 kb and it is interrupted by
three introns. All 11 ***zinc*** ***finger*** ***DNA*** -
binding domains are contiguously encoded within the last 3' exon.
The genomic region surrounding HF.10 exon 1 contains a ***CpG***
island and acts as a promoter in vitro. Using transient CAT assay in
cotransfection experiments in cultured cells, we have determined that the
HF.10 finger protein is a transcriptional transactivator. Restriction
enzyme mapping and partial nucleotide sequencing of the HF.10 pseudogene
indicated that it has arisen by retroposition of spliced HF.10 mRNA. In
situ hybridization experiments revealed that both the functional locus and
the pseudogene map to chromosome 3p21p22, a region that is frequently
deleted in small cell lung and renal carcinomas. Hybridization of the
HF.10 gene and the HF.10 pseudogene DNA probes to metaphases from a small
cell lung carcinoma cell line with the 3p deletion revealed that both loci
are part of the deleted chromosome region.

=> s l15 and methyltransferase#/ab,bi

11595 METHYLTRANSFERASE#/BI
5335587 AB/FA
5689 METHYLTRANSFERASE#/AB
(METHYLTRANSFERASE#/BI (L) AB/FA)
11595 METHYLTRANSFERASE#/BI
L17 1 L15 AND METHYLTRANSFERASE#/AB,BI

=> d bib ab

L17 ANSWER 1 OF 1 MEDLINE
AN 2000090946 MEDLINE
DN 20090946
TI Engineered zinc finger proteins that respond to DNA modification by HaeIII
and HhaI ***methyltransferase*** enzymes.
AU Isalan M; Choo Y
CS Laboratory of Molecular Biology, Medical Research Council, Hills Road,
Cambridge, CB2 2QH, UK.
SO JOURNAL OF MOLECULAR BIOLOGY, (2000 Jan 21) 295 (3) 471-7.
Journal code: J6V. ISSN: 0022-2836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 200004
EW 20000404
AB Zinc finger modules are capable of specifically interacting with DNA that
contains 5-methylcytosine (5-mC) in place of cytosine, suggesting that
zinc ***finger*** - ***DNA*** ***binding*** could be
regulated by extrinsic methylation of DNA. Here, we have used phage
display to engineer zinc finger proteins that detect and discriminate DNA
methylation by the prokaryotic enzymes HaeIII and HhaI. In these systems,
zinc finger-DNA complexes are induced by DNA modification using the
appropriate enzyme, which can therefore act as a switch. To further
develop the specificity of the switch, zinc finger discrimination between
5-mC and thymine in DNA sequences is demonstrated despite the presence of
the characteristic major groove methyl group that is common to both bases.

Specificity was achieved using a DNA-binding strategy involving synergy
between adjacent zinc fingers. We propose that engineered zinc fingers
that recognise particular DNA modifications, such as sequence-specific DNA
methylation, could be integrated into artificial regulatory circuits for
the control of gene expression and other biological processes.
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=> e isalan m/au

E1 22 ISAL J P/AU
E2 1 ISALA E/AU
E3 3 --> ISALAN M/AU
E4 1 ISALBERTI C/AU
E5 3 ISALBERTI L/AU
E6 1 ISALBERTI M/AU
E7 14 ISALES C/AU
E8 38 ISALES C M/AU
E9 2 ISALES FORSYTHE C M/AU
E10 1 ISALES L M/AU
E11 2 ISALGUE A M/AU
E12 1 ISALGUE M/AU

=> file medline embase biosis inpadoc caplus

COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 1.58 30.27

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
SINCE FILE TOTAL
ENTRY SESSION
CA SUBSCRIBER PRICE 0.00 -0.56

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FILE 'INPADOC' ENTERED AT 18:19:03 ON 22 JUN 2000
COPYRIGHT (C) 2000 European Patent Office, Vienna (EPO)

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=> s l17

'AB' IS NOT A VALID FIELD CODE
L18 3 L17

=> dup rem l18

PROCESSING COMPLETED FOR L18
L19 1 DUP REM L18 (2 DUPLICATES REMOVED)

=> d bib ab

L19 ANSWER 1 OF 1 MEDLINE DUPLICATE 1
AN 2000090946 MEDLINE
DN 20090946
TI Engineered zinc finger proteins that respond to DNA modification by HaeIII
and HhaI ***methyltransferase*** enzymes.
AU Isalan M; Choo Y
CS Laboratory of Molecular Biology, Medical Research Council, Hills Road,
Cambridge, CB2 2QH, UK.
SO JOURNAL OF MOLECULAR BIOLOGY, (2000 Jan 21) 295 (3) 471-7.
Journal code: J6V. ISSN: 0022-2836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 200004
EW 20000404
AB Zinc finger modules are capable of specifically interacting with DNA that
contains 5-methylcytosine (5-mC) in place of cytosine, suggesting that
zinc ***finger*** - ***DNA*** ***binding*** could be
regulated by extrinsic methylation of DNA. Here, we have used phage
display to engineer zinc finger proteins that detect and discriminate DNA
methylation by the prokaryotic enzymes HaeIII and HhaI. In these systems,
zinc finger-DNA complexes are induced by DNA modification using the
appropriate enzyme, which can therefore act as a switch. To further
develop the specificity of the switch, zinc finger discrimination between
5-mC and thymine in DNA sequences is demonstrated despite the presence of
the characteristic major groove methyl group that is common to both bases.
Specificity was achieved using a DNA-binding strategy involving synergy
between adjacent zinc fingers. We propose that engineered zinc fingers
that recognise particular DNA modifications, such as sequence-specific DNA
methylation, could be integrated into artificial regulatory circuits for
the control of gene expression and other biological processes.
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=> e bestor timothy/au

E1 98 BESTOR T H/AU
E2 2 BESTOR TIM/AU
E3 10 --> BESTOR TIMOTHY/AU
E4 74 BESTOR TIMOTHY H/AU
E5 1 BESTOR TIMOTHY HENRY/AU
E6 3 BESTOSO JOHN T/AU
E7 1 BESTOSO W J/AU
E8 3 BESTOUGEFF ALEXANDRE/AU
E9 5 BESTOUGEFF M/AU
E10 6 BESTOUGEFF M A/AU
E11 3 BESTOUGEFF MICHEL/AU
E12 5 BESTOUGEFF MICHEL A/AU

=> s e1-e5

L20 185 ("BESTOR T H"/AU OR "BESTOR TIM"/AU OR "BESTOR TIMOTHY"/AU OR
"BESTOR TIMOTHY H"/AU OR "BESTOR TIMOTHY HENRY"/AU)

=> s l20 and (chimeric or chimera)/ab,bi

'AB' IS NOT A VALID FIELD CODE
L21 5 L20 AND (CHIMERIC OR CHIMERA)/AB,BI

=> dup rem l21

PROCESSING COMPLETED FOR L21
L22 4 DUP REM L21 (1 DUPLICATE REMOVED)

=> d 1- bib ab

YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L22 ANSWER 1 OF 4 INPADOC COPYRIGHT 2000 EPO DUPLICATE 1

LEVEL 1
AN 42035735 INPADOC UP 20000502 UW 200017
TI ***CHIMERIC*** DNA-BINDING/DNA METHYLTRANSFERASE NUCLEIC ACID AND
POLYPEPTIDE AND USES THEREOF
IN BESTOR, TIMOTHY, H.
INS ***BESTOR TIMOTHY H***
INA US

PA THE TRUSTEES OF COLUMBIA UNIVERSITY IN THE CITY OF; BESTOR, TIMOTHY, H.
PAS UNIV COLUMBIA; BESTOR TIMOTHY H
PAA US; US
TL English; French
LA English
DT Patent
PIT WOA1 PUBL.OF THE INT.APPL. WITH INT.SEARCH REPORT
PI WO 9711972 A1 19970403
DS RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP MX US US
AI WO 1996-US15576 A 19960927
PRAI US 1995-4445 A2 19950928
US 1996-594866 A2 19960131
OSDW 97-212856
AB The present invention provides a ***chimeric*** protein which
comprises a mutated DNA methyltransferase portion and a DNA binding
protein portion that binds sufficiently close to a promoter sequence of a
target gene which promoter sequence contains a methylation site, to
specifically methylate the site and inhibit activity of the promoter and
thus inhibit expression of the target gene. This invention also provides
for a method for inhibiting the expression of a target gene which
includes contacting a promoter of the target gene with the
chimeric protein, so as to specifically methylate the promoter
sequence of the target gene thus inhibiting expression of the target
gene.

L22 ANSWER 2 OF 4 INPADOC COPYRIGHT 2000 EPO

LEVEL 1
AN 12181505 INPADOC
TI ***CHIMERIC*** DNA-BINDING/DNA METHYLTRANSFERASE NUCLEIC ACID AND
POLYPEPTIDE AND USES THEREOF
IN TIMOTHY H. BESTOR
INS ***BESTOR TIMOTHY H***
PA THE TRUSTEES OF COLUMBIA UNIVERSITY IN THE CITY OF NEW YORK
PAS UNIV COLUMBIA
DT Patent
PIT AUA1 COMP. SPEC. OPEN TO PUB. INSP.
PI AU 9673781 A1 19970417
AI AU 1996-73781 A 19960927
PRAI US 1995-4445 P 19950928
US 1996-594866 A 19960131
WO 1996-US15576 W 19960927

L22 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2000 ACS

AN 1997:791788 CAPLUS
DN 128:111239
TI Cytosine methylation targeted to pre-determined sequences
AU Xu, Guo-Liang; ***Bestor, Timothy H.***
CS Department of Genetics and Development, College of Physicians and Surgeons
of Columbia University, New York, NY, 10032, USA
SO Nat. Genet. (1997), 17(4), 376-378
CODEN: NGENEC; ISSN: 1061-4036
PB Nature America
DT Journal
LA English
AB Predetd. sequence specificities have now been conferred upon a DNA
methyltransferase by fusion to zinc-finger proteins. The sequence
specificity of zinc-finger proteins can be modified to direct cytosine
methylation to the promoters of target genes. Targeted methylation is
proposed as a new method for selective gene inactivation that stimulates
an existing biol. response.

L22 ANSWER 4 OF 4 MEDLINE
AN 92298390 MEDLINE
DN 92298390
TI Targeted mutation of the DNA methyltransferase gene results in embryonic
lethality.
AU Li E; ***Bestor T H*** ; Jaenisch R
CS Whitehead Institute for Biomedical Research, Cambridge, Massachusetts
02142..
NC GM43565 (NIGMS)
R35 CA 44339-05 (NCI)
SO CELL, (1992 Jun 12) 69 (6) 915-26.
Journal code: CQ4. ISSN: 0092-8674.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199209
AB Gene targeting in embryonic stem (ES) cells has been used to mutate the
murine DNA methyltransferase gene. ES cell lines homozygous for the
mutation were generated by consecutive targeting of both wild-type
alleles; the mutant cells were viable and showed no obvious abnormalities
with respect to growth rate or morphology, and had only trace levels of
DNA methyltransferase activity. A quantitative end-labeling assay showed
that the level of m5C in the DNA of homozygous mutant cells was about
one-third that of wild-type cells, and Southern blot analysis after
cleavage of the DNA with a methylation-sensitive restriction endonuclease
revealed substantial demethylation of endogenous retroviral DNA. The
mutation was introduced into the germline of mice and found to cause a
recessive lethal phenotype. Homozygous embryos were stunted, delayed in
development, and did not survive past mid-gestation. The DNA of homozygous
embryos showed a reduction of the level of m5C similar to that of
homozygous ES cells. These results indicate that while a 3-fold reduction
in levels of genomic m5C has no detectable effect on the viability or
proliferation of ES cells in culture, a similar reduction of DNA
methylation in embryos causes abnormal development and embryonic
lethality.

=> e isalan m/au

E1 1 ISALACO G/AU
E2 1 ISALACO S J/AU
E3 5 --> ISALAN M/AU
E4 24 ISALAN MARK/AU
E5 5 ISALBERTI C/AU
E6 1 ISALBERTI G/AU
E7 4 ISALBERTI L/AU
E8 5 ISALBERTI LORENZO/AU
E9 2 ISALBERTI M/AU
E10 1 ISALBERTI NATALE/AU
E11 1 ISALC MANIU A/AU
E12 1 ISALC MANIU ALEXANDRU/AU

=> s e3-e4

L23 29 ("ISALAN M"/AU OR "ISALAN MARK"/AU)

=> s l23 and (chimera or chimeric or fusion)/ab,bi

'AB' IS NOT A VALID FIELD CODE
L24 0 L23 AND (CHIMERA OR CHIMERIC OR FUSION)/AB,BI

=> s l23 and methyltransferase#/ab,bi

'AB' IS NOT A VALID FIELD CODE
L25 3 L23 AND METHYLTRANSFERASE#/AB,BI

=> dup rem l25

PROCESSING COMPLETED FOR L25
L26 1 DUP REM L25 (2 DUPLICATES REMOVED)

=> d bib ab

L26 ANSWER 1 OF 1 MEDLINE DUPLICATE 1
AN 2000090946 MEDLINE
DN 20090946
TI Engineered zinc finger proteins that respond to DNA modification by HaeIII
and HhaI ***methyltransferase*** enzymes.
AU ***Isalan M*** ; Choo Y
CS Laboratory of Molecular Biology, Medical Research Council, Hills Road,
Cambridge, CB2 2QH, UK.
SO JOURNAL OF MOLECULAR BIOLOGY, (2000 Jan 21) 295 (3) 471-7.
Journal code: J6V. ISSN: 0022-2836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 200004
EW 20000404
AB Zinc finger modules are capable of specifically interacting with DNA that
contains 5-methylcytosine (5-mC) in place of cytosine, suggesting that
zinc finger-DNA binding could be regulated by extrinsic methylation of
DNA. Here, we have used phage display to engineer zinc finger proteins
that detect and discriminate DNA methylation by the prokaryotic enzymes
HaeIII and HhaI. In these systems, zinc finger-DNA complexes are induced
by DNA modification using the appropriate enzyme, which can therefore act
as a switch. To further develop the specificity of the switch, zinc finger
discrimination between 5-mC and thymine in DNA sequences is demonstrated
despite the presence of the characteristic major groove methyl group that
is common to both bases. Specificity was achieved using a DNA-binding
strategy involving synergy between adjacent zinc fingers. We propose that
engineered zinc fingers that recognise particular DNA modifications, such
as sequence-specific DNA methylation, could be integrated into artificial
regulatory circuits for the control of gene expression and other
biological processes. Copyright 2000 Academic Press.

=> e choo y/au

E1 1 CHOO WOONG YONGO/AU
E2 15 CHOO WUNG YONG/AU
E3 30 --> CHOO Y/AU
E4 17 CHOO Y B/AU
E5 105 CHOO Y C/AU
E6 3 CHOO Y D/AU
E7 23 CHOO Y E/AU
E8 1 CHOO Y F/AU
E9 3 CHOO Y G/AU
E10 3 CHOO Y H/AU
E11 12 CHOO Y K/AU
E12 31 CHOO Y M/AU

=> s e1-e12

L27 243 ("CHOO WOONG YONGO"/AU OR "CHOO WUNG YONG"/AU OR "CHOO Y"/AU OR
"CHOO Y B"/AU OR "CHOO Y C"/AU OR "CHOO Y D"/AU OR "CHOO Y E"/AU
OR "CHOO Y F"/AU OR "CHOO Y G"/AU OR "CHOO Y H"/AU OR "CHOO Y
K"/AU OR "CHOO Y M"/AU)

=> e choo y m/au

E1 3 CHOO Y H/AU
E2 12 CHOO Y K/AU
E3 31 --> CHOO Y M/AU
E4 1 CHOO Y N/AU
E5 21 CHOO Y S/AU
E6 1 CHOO Y Y/AU
E7 1 CHOO YAW/AU
E8 2 CHOO YAW ANG/AU
E9 53 CHOO YEN/AU
E10 1 CHOO YEN MAY/AU
E11 1 CHOO YEOH SWEE/AU
E12 1 CHOO YEON CHUL/AU

=> s e4-e12

L28 82 ("CHOO Y N"/AU OR "CHOO Y S"/AU OR "CHOO Y Y"/AU OR "CHOO YAW"/A
U OR "CHOO YAW ANG"/AU OR "CHOO YEN"/AU OR "CHOO YEN MAY"/AU OR
"CHOO YEOH SWEE"/AU OR "CHOO YEON CHUL"/AU)

=> e choo yeon chul/au

E1 1 CHOO YEN MAY/AU
E2 1 CHOO YEOH SWEE/AU
E3 1 --> CHOO YEON CHUL/AU
E4 3 CHOO YEON DAE/AU
E5 6 CHOO YEON SIK/AU
E6 1 CHOO YEUN CHOL/AU
E7 1 CHOO YEUN DAE/AU
E8 1 CHOO YEUN DAI/AU
E9 7 CHOO YEUN MUN/AU
E10 1 CHOO YEUN SOO/AU
E11 1 CHOO YEW KUAN/AU
E12 4 CHOO YIN YEOW/AU

=> s e9

L29 7 "CHOO YEUN MUN"/AU

=> s l27 or l28

L30 325 L27 OR L28

=> s l30 and methyltransferase#/ab,bi

'AB' IS NOT A VALID FIELD CODE
L31 3 L30 AND METHYLTRANSFERASE#/AB,BI

=> dup rem l31

PROCESSING COMPLETED FOR L31
L32 1 DUP REM L31 (2 DUPLICATES REMOVED)

=> d

L32 ANSWER 1 OF 1 MEDLINE DUPLICATE 1
AN 2000090946 MEDLINE
DN 20090946
TI Engineered zinc finger proteins that respond to DNA modification by HaeIII
and HhaI ***methyltransferase*** enzymes.
AU Isalan M; ***Choo Y***
CS Laboratory of Molecular Biology, Medical Research Council, Hills Road,
Cambridge, CB2 2QH, UK.
SO JOURNAL OF MOLECULAR BIOLOGY, (2000 Jan 21) 295 (3) 471-7.
Journal code: J6V. ISSN: 0022-2836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 200004
EW 20000404

=> d his

(FILE 'HOME' ENTERED AT 18:09:14 ON 22 JUN 2000)

FILE 'MEDLINE' ENTERED AT 18:09:28 ON 22 JUN 2000
L1 2136 S ZINC FINGER AND DNA BINDING/AB,BI
L2 87 S L1 AND POLYPEPTIDE/AB,BI
L3 0 S L2 AND METHYLTRANSFERASE/AB,BI
L4 3392 S CPG/AB,BI
L5 209 S L4 AND METHYLTRANSFERASE/AB,BI
L6 6 S L5 AND ZINC/AB,BI
L7 12 S CPG-SPECIFIC/AB,BI
L8 2 S L7 AND METHYLTRANSFERASE#/AB,BI
L9 22158 S CHIMERIC OR CHIMERA/AB,BI

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L10       114 S L9 AND ZINC-FINGER/AB,BI
L11        83 S L10 AND DNA BINDING/AB,BI
L12         1 S L11 AND CPG?/AB,BI

   FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS'
ENTERED AT 18:14:34 ON 22
   JUN 2000
L13         2 S L12
L14         2 DUP REM L13 (0 DUPLICATES REMOVED)

   FILE 'MEDLINE' ENTERED AT 18:16:29 ON 22 JUN 2000
L15       217 S ZINC-FINGER BINDING OR ZINC-FINGER DNA
BINDING/AB,BI
L16         1 S L15 AND CPG?/AB,BI
L17         1 S L15 AND METHYLTRANSFERASE#/AB,BI
            E ISALAN M/AU

   FILE 'MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS'
ENTERED AT 18:19:03 ON 22
   JUN 2000
L18         3 S L17
L19         1 DUP REM L18 (2 DUPLICATES REMOVED)
            E BESTOR TIMOTHY/AU
L20       185 S E1-E5
L21         5 S L20 AND (CHIMERIC OR CHIMERA)/AB,BI
L22         4 DUP REM L21 (1 DUPLICATE REMOVED)
            E ISALAN M/AU
L23        29 S E3-E4
L24         0 S L23 AND (CHIMERA OR CHIMERIC OR
FUSION)/AB,BI
L25         3 S L23 AND METHYLTRANSFERASE#/AB,BI
L26         1 DUP REM L25 (2 DUPLICATES REMOVED)
            E CHOO Y/AU
L27       243 S E1-E12
            E CHOO Y M/AU
L28        82 S E4-E12
            E CHOO YEON CHUL/AU
L29         7 S E9
L30       325 S L27 OR L28
L31         3 S L30 AND METHYLTRANSFERASE#/AB,BI
L32         1 DUP REM L31 (2 DUPLICATES REMOVED)

=> log y

COST IN U.S. DOLLARS                SINCE FILE     TOTAL
                                    ENTRY   SESSION
FULL ESTIMATED COST                   91.73    122.00

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
SINCE FILE     TOTAL
                                    ENTRY   SESSION
CA SUBSCRIBER PRICE                   -0.56     -1.12

STN INTERNATIONAL LOGOFF AT 18:25:05 ON 22 JUN 2000
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